



Case Report

A fish-stunning wound infection with acute cardiac injury

Abstract

Vibrio vulnificus typically causes septicemia and necrotic wound infection. Among *V vulnificus*-related complications, acute nonthrombotic myocardial damage has not been reported. The most effective antimicrobial treatment of *V vulnificus* infection includes combination of a third-generation cephalosporin and a tetracycline or its analogue. However, recommendations of a fourth-generation cephalosporin-based regimen for treating the disease are not established. A 67-year-old diabetic man acquired *V vulnificus* infection via a fish-stunning wound on the right foot. The patient developed septicemia and hemorrhagic bullous necrotic wounds and followed by acute nonthrombotic cardiac injury with low cardiac output. After initial resuscitation, we applied dobutamine inotropic therapy with combination of ceftiofime and ciprofloxacin or minocycline, which achieved a good clinical outcome.

Vibrio vulnificus infection is the leading cause of death related to seafood consumption in the patients particularly with chronic liver disease after consuming raw oyster or when an open wound is exposed to seawater with high concentrations of *V vulnificus* [1–3]. It typically causes septicemia and necrotic wound infection. However, the complication of acute nonthrombotic myocardial damage has not been reported. Besides, recommendations of a fourth-generation cephalosporin to treat the disease are not established.

A 67-year-old diabetic man was brought to the emergency department because of painful swelling of the right leg for 3 days. The patient had exposure to seawater, and some fish stung his right foot 4 days ago. A fish-stunning hole could be seen on the dorsal aspect of the right foot wound with hemorrhagic bullae (Fig.). The blood pressure was ever dropped to 65/51 mm Hg.

Laboratory data revealed the following: white blood cell count, 6500/μL; hemoglobin level, 14 g/dL; hematocrit, 39.1%; platelet count, 53 000/μL; serum Na, 122.9 mEq/L; K, 3.22 mEq/L; blood sugar, 239 mg/dL; lactate level, 3.5 mmol/L; blood urine nitrogen, 23 mg/dL; creatinine, 1.1 mg/dL; serum glutamic oxaloacetic transaminase, 59 IU/L; and serum glutamic pyruvic transaminase, 29 IU/L. The initial central venous oxygen saturation (ScvO₂) was 78.3%. The base excess of initial arterial blood glass analysis was −5.2 mmol/L. Antimicrobial therapy was given with ceftiofime 2 g every 8 hours plus ciprofloxacin 400 mg every 12 hours intravenously.

After initial resuscitation with 3950 mL fluid on day 1, the patient still needed norepinephrine to keep an adequate mean arterial pressure. On day 2, however, troponin I level was elevated to 7.06 ng/mL accompanied with cold feet temperature (28°C). The serum lactate level remained elevated (3.5 mmol/L), and the arterial blood base excess became −11.1 mmol/L. Dobutamine was initiated because the ScvO₂ dropped to 50%, suspecting low cardiac output.

Electrocardiography did not show significant ST-T changes in all leads. Echocardiography showed adequate left ventricle performance without abnormality of global and regional wall motion. On day 3, norepinephrine was discontinued. Central venous oxygen saturation increased to 74%, lactate dropped to 1.5 mmol/L, troponin I became 2.81 ng/mL, and base excess recovered to −0.1 mmol/L. The patient underwent minimal fasciotomy and debridement for the necrotic tissue. Both the blood and wound cultures yielded *V vulnificus*, which was susceptible to minocycline, ciprofloxacin, ceftazidime, and ceftiofime. Antimicrobial therapy was revised to ceftiofime 2 g every 8 hours plus minocycline 100 mg every 12 hours intravenously, and he was discharged uneventfully on day 11. The wound *Mycobacterium* culture yielded no growth at last.

The etiology causing hemorrhagic bullous cutaneous lesions includes *V vulnificus*, *Staphylococcus aureus*, *Streptococcus pyogenes*, and *Mycobacterium marinum*. *V vulnificus* is a species of halophilic gram-negative bacterium that is mostly recovered from marine environments [1]. *V vulnificus* infection typically causes primary septicemia and necrotic wound infection. Most patients develop severe sepsis and cellulitis with rapid formation of hemorrhagic bullae, which would progress to necrotizing fasciitis in severe cases [2]. Case-fatality rates are greater than 50% for *V vulnificus* septicemia and about 15% for wound infections, mostly in patients with septic shock and rapid death within 2 days of admission [2,3]. The most effective antibiotic treatment of *V vulnificus* infection includes combination of a third-generation cephalosporin and a tetracycline, doxycycline, or minocycline [3–5]. To our knowledge, treatment with a fourth-generation cephalosporin-based regimen such as ceftiofime or ceftiofime for *V vulnificus* infection has not been reported in the English literature. A combination of a fourth-generation cephalosporin (eg, ceftiofime) and minocycline might be optimal for such lesions suspicious of *V vulnificus* infection with more extended coverage of gram-positive organisms at empirical stage. Besides, ciprofloxacin has good in vitro and in vivo activities against the *V vulnificus* isolates tested in Taiwan [6].

Acute cardiac injury by septic cardiomyopathy or nonthrombotic acute coronary syndrome with myocardial damage is frequently observed in patients with severe sepsis and septic shock [7,8]. However, *V vulnificus* infection-related acute cardiac injury has not been reported in the literature. In 2008 Surviving Sepsis Campaign, dobutamine inotropic therapy has been suggested when the cardiac output of a septic patient remains low despite initiate fluid resuscitation [9]. Our patient experienced unexpected low ScvO₂ associated with elevated troponin I and lactate levels after initial fluid resuscitation, which might imply acute cardiac injury with low-cardiac-output syndrome. The normal cardiac wall motion on echocardiography did not favor the possibility of thrombotic coronary artery syndrome. After dobutamine inotropic therapy, our patient



Fig. Hemorrhagic bullous necrotic wound lesions on the right foot with a fish-stunning wound.

responded well with recovery of $ScvO_2$ to normal level, increasing daily urine output, and decreasing serum lactate level.

In conclusion, we demonstrated the clinical effectiveness of dobutamine, cefpirome, and minocycline for treating *V. vulnificus* necrotizing wound infection complicated with acute cardiac injury. A fish-stunning wound should remind physicians that the wound was infected by *V. vulnificus*. Acute *V. vulnificus*-related nonthrombotic myocardial damage has not been reported yet.

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References

- [1] Hollis DG, Weaver RE, Baker CN, Thornsberry C. Halophilic *Vibrio* species isolated from blood cultures. *J Clin Microbiol* 1976;3:425–31.
- [2] Klontz KC, Lieb S, Schreiber M, et al. Syndromes of *Vibrio vulnificus* infections. Clinical and epidemiologic features in Florida cases, 1981–1987. *Ann Intern Med* 1988;109:318–23.
- [3] Chuang YC, Yuan CY, Liu CY, et al. *Vibrio vulnificus* infection in Taiwan: report of 28 cases and review of clinical manifestations and treatment. *Clin Infect Dis* 1992;15:271–6.
- [4] Liu JW, Lee IK, Tang HJ, et al. Prognostic factors and antibiotics in *Vibrio vulnificus* septicemia. *Arch Intern Med* 2006;166:2117–23.
- [5] Chen SC, Lee YT, Tsai SJ, et al. Antibiotic therapy for necrotizing fasciitis caused by *Vibrio vulnificus*: retrospective analysis of an 8 year period. *J Antimicrob Chemother* 2012;67:488–93.
- [6] Tang HJ, Chang MC, Ko WC, et al. In vitro and in vivo activities of newer fluoroquinolones against *Vibrio vulnificus*. *Antimicrob Agents Chemother* 2002;46:3580–4.
- [7] Kalla C, Raveh D, Algur N, et al. Incidence and significance of a positive troponin test in bacteremic patients without acute coronary syndrome. *Am J Med* 2008;121:909–15.
- [8] Altmann DR, Korte W, Maeder MT, et al. Elevated cardiac troponin I in sepsis and septic shock: no evidence for thrombus associated myocardial necrosis. *PLoS One* 2010;5:e9017.
- [9] Dellinger RP, Levy MM, Carlet JM, et al. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock: 2008. *Crit Care Med* 2008;36:296–327.